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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Dec 17	The CA Lexicon available in the CAPLUS and CA files
NEWS 3	Feb 06	Engineering Information Encompass files have new names
NEWS 4	Feb 16	TOXLINE no longer being updated
NEWS 5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS 6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS 7	May 07	DGENE Reload
NEWS 8	Jun 20	Published patent applications (A1) are now in USPATFULL
NEWS 9	JUL 13	New SDI alert frequency now available in Derwent's DWPI and DPCI
NEWS 10	Aug 23	In-process records and more frequent updates now in MEDLINE
NEWS 11	Aug 23	PAGE IMAGES FOR 1947-1966 RECORDS IN CAPLUS AND CA
NEWS 12	Aug 23	Adis Newsletters (ADISNEWS) now available on STN
NEWS 13	Sep 17	IMSworld Pharmaceutical Company Directory name change to PHARMASEARCH
NEWS 14	Oct 09	Korean abstracts now included in Derwent World Patents Index
NEWS 15	Oct 09	Number of Derwent World Patents Index updates increased
NEWS 16	Oct 15	Calculated properties now in the REGISTRY/ZREGISTRY File
NEWS 17	Oct 22	Over 1 million reactions added to CASREACT
NEWS 18	Oct 22	DGENE GETSIM has been improved
NEWS 19	Oct 29	AAASD no longer available
NEWS 20	Nov 19	New Search Capabilities USPATFULL and USPAT2
NEWS 21	Nov 19	TOXCENTER(SM) - new toxicology file now available on STN
NEWS 22	Nov 29	COPPERLIT now available on STN
NEWS 23	Nov 29	DWPI revisions to NTIS and US Provisional Numbers
NEWS 24	Nov 30	Files VETU and VETB to have open access
NEWS 25	Dec 10	WPINDEX/WPIDS/WPIX New and Revised Manual Codes for 2002
NEWS 26	Dec 10	DGENE BLAST Homology Search
NEWS 27	Dec 17	WELDASEARCH now available on STN
NEWS 28	Dec 17	STANDARDS now available on STN
NEWS 29	Dec 17	New fields for DPCI
NEWS 30	Dec 19	CAS Roles modified
NEWS 31	Dec 19	1907-1946 data and page images added to CA and CAplus

NEWS EXPRESS	August 15 CURRENT WINDOWS VERSION IS V6.0c, CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP), AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001
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FILE 'HOME' ENTERED AT 09:06:19 ON 03 JAN 2002

=> file medline biosis embase caplus uspatfull

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FILE 'MEDLINE' ENTERED AT 09:06:29 ON 03 JAN 2002

FILE 'BIOSIS' ENTERED AT 09:06:29 ON 03 JAN 2002
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FILE 'USPATFULL' ENTERED AT 09:06:29 ON 03 JAN 2002
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=> s steroid (p) receptor (p) fluorescence (p) bind

L1 45 STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND

=> s estrogen (p) receptor (p) fluorescence (p) bind

L2 79 ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND

=> s estrogen (p) receptor (p) fluorescence (p) bind (p) polarization

L3 8 ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZATION

=> dup rem l3

PROCESSING COMPLETED FOR L3
L4 3 DUP REM L3 (5 DUPLICATES REMOVED)

=> d l4 total ibib kwic

L4 ANSWER 1 OF 3 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001374733 MEDLINE
DOCUMENT NUMBER: 21324553 PubMed ID: 11431146
TITLE: Interactions of synthetic estrogens with human estrogen receptors.
AUTHOR: Nikov G N; Eshete M; Rajnarayanan R V; Alworth W L
CORPORATE SOURCE: Department of Chemistry, Tulane University, New Orleans,

Louisiana 70118, USA.
SOURCE: JOURNAL OF ENDOCRINOLOGY, (200 Jul) 170 (1) 137-45.
Journal code: I1J; 0375363. ISSN: 0022-0795.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010820
Last Updated on STN: 20010820
Entered Medline: 20010816

AB Synthetic **estrogens** have diverse chemical structures and may either positively or negatively affect the estrogenic signaling pathways through interactions with the **estrogen receptors** (ERs). Modeling studies suggest that 4-(1-adamantyl)phenol (AdP) and 4,4'-(1,3-adamantanediyl)diphenol (AdDP) can **bind** in the ligand binding site of ERalpha. We used **fluorescence polarization** (FP) to compare the binding affinities of AdP, AdDP and 2-(1-adamantyl)-4-methylphenol (AdMP) for human ERalpha and ERbeta with the binding. . . (4OHT). Competition binding experiments show that AdDP has greater affinity for both ERs than does AdP, while AdMP does not **bind** the **receptor** proteins. The relative binding affinities of AdDP and AdP are weaker than the affinity of DES or 4OHT for both ERs with the exception of AdDP, which **binds** ERbeta with higher affinity than does 4OHT. We also found that AdDP and AdP cause differential conformational changes in ERalpha and ERbeta, which result in altered affinities of the ERs for fluorescein-labeled **estrogen** response elements (EREs) using a direct binding FP assay. The results show that ERbeta liganded with either AdDP or AdP. . .

L4 ANSWER 2 OF 3 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001058971 MEDLINE
DOCUMENT NUMBER: 20472537 PubMed ID: 11017892
TITLE: Interactions of dietary estrogens with human estrogen receptors and the effect on estrogen receptor-estrogen response element complex formation.
COMMENT: Comment in: Environ Health Perspect. 2000 Sep;108(9):A416
AUTHOR: Nikov G N; Hopkins N E; Boue S; Alworth W L
CORPORATE SOURCE: Department of Chemistry, Tulane University, New Orleans, Louisiana 07118, USA.
SOURCE: ENVIRONMENTAL HEALTH PERSPECTIVES, (2000 Sep) 108 (9) 867-72.
Journal code: EI0. ISSN: 0091-6765.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20001222

AB Epidemiologic and experimental studies support the hypothesis that dietary **estrogens** from plant sources (phytoestrogens) may play a role in the prevention of breast and prostate cancer. The molecular mechanisms for such chemopreventive effect are still unclear. We investigated the possibility that phytoestrogens may **bind** differentially to **estrogen receptor** proteins (ER[alpha] and ERss) and affect the interactions of the ligand-ER complexes with different **estrogen** response element (ERE) sequences. We used **fluorescence polarization** to measure the binding

affinities of genistein, coumestrol, daidzein, glyceollin, and zearalenone
for human ER[alpha] and ERss. Competition binding experiments. . . on the ability of ER[alpha] and ERss to associate with specific DNA sequences
(EREs). The direct binding of human recombinant **estrogen receptors** to fluorescein-labeled EREs indicates that phytoestrogens can cause conformational changes in both human ERs, which results in altered affinities of. . .

L4 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:112498 CAPLUS
DOCUMENT NUMBER: 128:176476
TITLE: A method for quantitating competitive binding of molecules to steroid hormone receptors utilizing fluorescence polarization
INVENTOR(S): Bolger, Randall E.; Ervin, Kerry M.; Lowery, Robert G.; Checovich, William J.
PATENT ASSIGNEE(S): Panvera Corp., USA; Burke, Thomas, J.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9805962	A1	19980212	WO 1997-US13538	19970801

RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1996-23034 19960802
AB The system comprises mixing a **fluorescence**-emitting compd. that **binds** to the steroid hormone **receptors**, particularly the **estrogen receptor**, in a soln. contg. the steroid hormone **receptors**. Then, measuring the **fluorescence polarization** of the soln. Subsequently, incubating the soln. with at least one mol. that may compete with the compd. for interaction with the steroid hormone **receptors**. Measuring the **fluorescence polarization** of the soln. again. Finally, comparing the **fluorescence polarization** measurements to quantify any competitive interaction. A **fluorescence** -emitting compd. such as a **fluorescence**-emitting hormone can be used in combination with a fluorophore covalently coupled to an oligonucleotide to study how hormone and oligonucleotide binding to the hormone **receptor** are affected by each other.
IT Pesticides
(detection of environmental compds. which **bind** **estrogen receptors** using a competitive **fluorescence polarization** assay)
IT 8001-35-2, Toxaphene 12789-03-6, Chlordane
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(detection of environmental compds. which **bind** **estrogen receptors** using a competitive **fluorescence polarization** assay)

=> d his

(FILE 'HOME' ENTERED AT 09:06:19 ON 03 JAN 2002)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 09:06:29 ON 03 JAN 2002
L1 45 S STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND
L2 79 S ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND

L3 8 S ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZAT
L4 3 DUP REM L3 (5 DUPLICATES REMOVED)

=> s steroid (p) receptor (p) fluorescence (p) bind (p) polarization

L5 5 STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZATION

=> dup rem l5

PROCESSING COMPLETED FOR L5
L6 2 DUP REM L5 (3 DUPLICATES REMOVED)

=> d l6 total ibib kwic

L6 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:112498 CAPLUS
DOCUMENT NUMBER: 128:176476
TITLE: A method for quantitating competitive binding of molecules to steroid hormone receptors utilizing fluorescence polarization
INVENTOR(S): Bolger, Randall E.; Ervin, Kerry M.; Lowery, Robert G.; Checovich, William J.
PATENT ASSIGNEE(S): Panvera Corp., USA; Burke, Thomas, J.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9805962	A1	19980212	WO 1997-US13538	19970801

RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1996-23034 19960802
AB The system comprises mixing a **fluorescence**-emitting compd. that **binds** to the **steroid** hormone **receptors**, particularly the estrogen **receptor**, in a soln. contg. the **steroid** hormone **receptors**. Then, measuring the **fluorescence polarization** of the soln. Subsequently, incubating the soln. with at least one mol. that may compete with the compd. for interaction with the **steroid** hormone **receptors**. Measuring the **fluorescence polarization** of the soln. again. Finally, comparing the **fluorescence polarization** measurements to quantify any competitive interaction. A **fluorescence**-emitting compd. such as a **fluorescence**-emitting hormone can be used in combination with a fluorophore covalently coupled to an oligonucleotide to study how hormone and oligonucleotide binding to the hormone **receptor** are affected by each other.

L6 ANSWER 2 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 86221226 MEDLINE
DOCUMENT NUMBER: 86221226 PubMed ID: 3011559
TITLE: Sex steroid and prostaglandin interactions upon the purified rat myometrial plasma membranes.
AUTHOR: Deliconstantinos G; Fotiou S
SOURCE: MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1986 May) 45 (2-3) 149-56.
Journal code: E69; 7500844. ISSN: 0303-7207.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 198607
ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19860710

AB . . . with myometrial plasma membranes (MPM) at a concentration of 1 X 10(-6) M for 1 h at 37 degrees C, **bind** into MPM at pmolar concentrations. Unlabeled **steroids** inhibited [3H]PGE2 and [3H]PGF2 alpha binding to MPM in a dose-dependent manner. Membrane-bound and free **steroids** or PGs were found to be essentially unchanged under the present incubation conditions. Ca2+ ions up to 10 mM increased **steroid** binding into MPM. Molecular interactions between **steroids** and MPM were assessed by measuring the steady-state **fluorescence polarization** of 1,6-diphenyl-1,3,5-hexatriene (DPH), and by estimating the changes in the allosteric properties of MPM-bound (Na+ + K+)ATPase by fluoride (F-). **Steroids** appear to increase the MPM fluidity, evaluated through changes in the Hill coefficient for MPM-bound (Na+ + K+)ATPase by F- and by the **fluorescence polarization** method. Binding of sex **steroids** to MPM increased the membrane fluidity and decreased the binding of the uterus stimulatory PGs by membrane **receptors**. These studies provide a basis for postulating that a 'non-genomic' mechanism of sex **steroids** induces reduction of uterine contractions.

=> d his

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FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 09:06:29 ON 03 JAN 2002

L1 45 S STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND
L2 79 S ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND
L3 8 S ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZAT
L4 3 DUP REM L3 (5 DUPLICATES REMOVED)
L5 5 S STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZATI
L6 2 DUP REM L5 (3 DUPLICATES REMOVED)

=> s steroid (p) receptor (p) fluorescence (p) bind (p) dna

L7 5 STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) DNA

=> dup rem l7

PROCESSING COMPLETED FOR L7
L8 2 DUP REM L7 (3 DUPLICATES REMOVED)

=> d l8 total ibib kwic

L8 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:476304 CAPLUS
DOCUMENT NUMBER: 127:105220
TITLE: Monitoring DNA binding molecules in living cells containing a steroid receptor response element array using a fluorescent chimeric protein of the steroid receptor
INVENTOR(S): Htun, Han; Hager, Gordon L.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA;
Htun, Han; Hager, Gordon L.
SOURCE: PCT Int. Appl., 99 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9720931	A1	19970612	WO 1996-US19516	19961206
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2239951	AA	19970612	CA 1996-2239951	19961206
AU 9712834	A1	19970627	AU 1997-12834	19961206

PRIORITY APPLN. INFO.: US 1995-8373 P 19951208
WO 1996-US19516 W 19961206

AB A method of screening for a compd. that **binds** to a selected nucleic acid is provided that comprises contacting a compd. fluorescently labeled by a fluorescent protein with a cell having a plurality of copies of the nucleic acid in an array such that the nucleic acid can be directly detected when bound by fluorescently labeled compd. The location of **fluorescence** within the cell is detected such that **fluorescence** aggregated at the site of the nucleic acid array indicates a compd. that **binds** to the selected nucleic acid. In particular compds. such a transcription factor can be screened. Reagents for such method are provided including a mammalian cell having a plurality of **steroid receptor** response elements in an array such that the response element can be directly detected when bound by fluorescently labeled **steroid receptor** and a chimeric protein comprising a fluorescent protein fused to a **steroid receptor**. Thus, a chimeric protein is constructed comprising a 27-kDa green fluorescent protein (GFP, from Aequorea victoria) and fused by a (Gly-Ala)5 peptide linker to the N-terminal second residue of rat glucocorticoid **receptor** (GR). Improved **fluorescence** is achieved by using a GFP variant contg. a serine-65 to threonine substitution, which increases the efficiency of formation of the GFP chromophore, and a GR variant contg. a cysteine-656 to glycine mutation has higher affinity for its ligand than endogenous **receptor**. A mammalian cell line named 3134 was derived by transfection of murine mammary carcinoma line C127 with a plasmid contg. 3 functional segments: (a) the bovine papilloma virus 69% transforming fragment serving as a replicon in mammalian cells; (b) mouse mammary tumor virus (MMTV) LTR is a **steroid** responsive promoter and contains the GR binding sites; and (c) the Ha-v-ras gene is a transforming oncogene and serves as a reporter for the MMTV promoter. The MMTV LTRs are organized in a head-to-tail tandem array of .apprx.200 copies, and since each promoter sequence contains 4 GR binding sites, the complete array contains 100 GR binding sites. This cell is used to visualize directly the interaction between the fluorescent, chimeric GR and its binding site in chromatin in living cells. The system allows screening for **DNA**-binding ligands that (1) activate gene targeting by **steroid receptor**, (2) activate translocation of the **steroid receptor** to the nucleus, (3) are antagonists or agonists of the **steroid receptor**.

L8 ANSWER 2 OF 2 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1998008920 MEDLINE
DOCUMENT NUMBER: 98008920 PubMed ID: 9344655

TITLE: Chromosomal mapping of the human and murine orphan receptors ERRalpha (ESRRA) and ERRbeta (ESRRB) and identification of a novel human ERRalpha-related pseudogene.
AUTHOR: Sladek R; Beatty B; Squire J; Copeland N G; Gilbert D J; Jenkins N A; Giguere V
CORPORATE SOURCE: Royal Victoria Hospital, Department of Biochemistry, McGill University, 687 Pine Avenue West, Montr-eal, Quebec, H3A 1A1, Canada.
SOURCE: GENOMICS, (1997 Oct 15) 45 (2) 320-6.
Journal code: GEN; 8800135. ISSN: 0888-7543.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF022222; GENBANK-U85258; GENBANK-X51416
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980224
Last Updated on STN: 19980224
Entered Medline: 19980212

AB The estrogen-related **receptors** ERRalpha and ERRbeta (formerly ERR1 and ERR2) form a subgroup of the **steroid**/thyroid/retinoid **receptor** family. ERRalpha and ERRbeta are homologous to the estrogen **receptor** and **bind** similar **DNA** targets; however, they are unable to activate gene transcription in response to estrogens. We have used interspecific backcross analysis to map the murine Estrra locus to chromosome 19 and Estrrb to mouse chromosome 12. Using **fluorescence** in situ hybridization, we have mapped the human ESRRA gene to chromosome 11q12-q13 and the human ESRRB gene to chromosome. . . . chromosome 13q12.1. To our knowledge, this represents the first report of a pseudogene associated with a member of the nuclear **receptor** superfamily.

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(FILE 'HOME' ENTERED AT 09:06:19 ON 03 JAN 2002)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS, USPATFULL' ENTERED AT 09:06:29 ON 03 JAN 2002
L1 45 S STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND
L2 79 S ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND
L3 8 S ESTROGEN (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZAT
L4 3 DUP REM L3 (5 DUPLICATES REMOVED)
L5 5 S STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) POLARIZATI
L6 2 DUP REM L5 (3 DUPLICATES REMOVED)
L7 5 S STEROID (P) RECEPTOR (P) FLUORESCENCE (P) BIND (P) DNA
L8 2 DUP REM L7 (3 DUPLICATES REMOVED)

=> dup rem l1

PROCESSING COMPLETED FOR L1
L9 18 DUP REM L1 (27 DUPLICATES REMOVED)

=> d l9 total ibib kwic

L9 ANSWER 1 OF 18 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2001246639 MEDLINE
DOCUMENT NUMBER: 21136238 PubMed ID: 11238589
TITLE: High constitutive glucocorticoid receptor beta in human

neutrophils enables them to reduce their spontaneous rate of cell death in response to glucocorticosteroids.

AUTHOR: Strickland I; Kisich K; Hauk P J; Vottero A; Chrousos G P; Klemm D J; Leung D Y

CORPORATE SOURCE: Department of Pediatrics, National Jewish Medical and Research Center, 1400 Jackson St., Denver, Colorado 80206, USA.

CONTRACT NUMBER: AR41256 (NIAMS)
HL34303 (NHLBI)
HL36577 (NHLBI)
HL37260 (NHLBI)

SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (2001 Mar 5) 193 (5) 585-94.
Journal code: I2V; 2985109R. ISSN: 0022-1007.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200105

ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010510

AB . . . neutrophil-mediated diseases. Development of new antiinflammatory strategies for such diseases would be aided by an understanding of mechanisms underlying differential **steroid** responsiveness. Two protein isoforms of the human glucocorticoid **receptor** (GR) exist, GRalpha and GRbeta, which arise from alternative splicing of the GR pre-mRNA primary transcripts. GRbeta does not **bind** glucocorticoids and is an inhibitor of GRalpha activity. Relative amounts of these two GRs can therefore determine the level of. . . human neutrophils and peripheral blood mononuclear cells (PBMCs) were studied to determine the relative amounts of each GR isoform.The mean **fluorescence** intensity (MFI) using immunofluorescence analysis for GRalpha was 475 +/- 62 and 985 +/- 107 for PBMCs and neutrophils, respectively.. . .

L9 ANSWER 2 OF 18 MEDLINE DUPLICATE 2

ACCESSION NUMBER: 2001454545 MEDLINE

DOCUMENT NUMBER: 21391675 PubMed ID: 11500849

TITLE: Multiplexed molecular interactions of nuclear receptors using fluorescent microspheres.

AUTHOR: Iannone M A; Consler T G; Pearce K H; Stimmel J B; Parks D J; Gray J G

CORPORATE SOURCE: Department of Gene Expression and Protein Biochemistry, GlaxoSmithKline, Research Triangle Park, North Carolina 27709-3398, USA.. mai49583@gsk.com

SOURCE: CYTOMETRY, (2001 Aug 1) 44 (4) 326-37.
Journal code: D92; 8102328. ISSN: 0196-4763.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200110

ENTRY DATE: Entered STN: 20010814
Last Updated on STN: 20011022
Entered Medline: 20011018

AB BACKGROUND: We describe a novel microsphere-based system to identify and characterize multiplexed interactions of nuclear **receptors** with peptides that represent the LXXLL binding region of coactivator proteins. METHODS: In this system, individual microsphere populations with unique. . . fluorescent profiles are coupled to specific coactivator peptides. The coactivator peptide-coupled microsphere populations are combined and incubated with a nuclear **receptor** that has been coupled to a

green fluorochrome. Flow cytometric analysis of the microspheres simultaneously decodes each population and detects the binding of **receptor** to respective coactivator peptides by the acquisition of green **fluorescence**. RESULTS: We have used this system to determine the binding affinities of human estrogen **receptor** beta ligand binding domain (ERbeta LBD) and human peroxisome proliferator activated **receptor** gamma ligand binding domain (PPARgamma LBD) to a set of 34 coactivator peptides. Binding of ERbeta LBD to a coactivator peptide sequence containing the second LXXLL motif of **steroid receptor** coactivator-1 (SRC-1(2) (676-700) is shown to be specific and saturable. Analysis of **receptor** binding to a multiplexed set of coactivator peptides shows PPARgamma LBD **binds** with high affinity to cAMP response element binding protein (CBP) peptides and to the related P300 peptide while ERbeta LBD. . . of antagonist (raloxifene or tamoxifen). CONCLUSIONS: This unique microsphere-based system is a sensitive and efficient method to simultaneously evaluate many **receptor**-coactivator interactions in a single assay volume. In addition, the system offers a powerful approach to study small molecule modulation of nuclear **receptor** binding.

L9 ANSWER 3 OF 18 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 2001288952 EMBASE

TITLE: Multiplexed molecular interactions of nuclear receptors using fluorescent microspheres.

AUTHOR: Iannone M.A.; Consler T.G.; Pearce K.H.; Stimmel J.B.; Parks D.J.; Gray J.G.

CORPORATE SOURCE: M.A. Iannone, GlaxoSmithKline, 5 Moore Drive, Res. Triangle Park, NC 27709-3398, United States. mai49583@gsk.com

SOURCE: Communications in Clinical Cytometry, (1 Aug 2001) 46/4 (326-337).
Refs: 36
ISSN: 0196-4763 CODEN: CCCYEM

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Background: We describe a novel microsphere-based system to identify and characterize multiplexed interactions of nuclear **receptors** with peptides that represent the LXXLL binding region of coactivator proteins. Methods: In this system, individual microsphere populations with unique. . . fluorescent profiles are coupled to specific coactivator peptides. The coactivator peptide-coupled microsphere populations are combined and incubated with a nuclear **receptor** that has been coupled to a green fluorochrome. Flow cytometric analysis of the microspheres simultaneously decodes each population and detects the binding of **receptor** to respective coactivator peptides by the acquisition of green **fluorescence**. Results: We have used this system to determine the binding affinities of human estrogen **receptor** .beta. ligand binding domain (ER.beta. LBD) and human peroxisome proliferator activated **receptor** .gamma. ligand binding domain (PPAR.gamma. LBD) to a set of 34 coactivator peptides. Binding of ER.beta. LBD to a coactivator peptide sequence containing the second LXXLL motif of **steroid receptor** coactivator-1 (SRC-1(2) (676-700) is shown to be specific and saturable. Analysis of **receptor** binding to a multiplexed set of coactivator peptides shows PPAR.gamma. LBD **binds** with high affinity to cAMP response element binding protein (CBP) peptides and to the related P300 peptide while ER.beta. LBD. . . of antagonist (raloxifene or tamoxifen). Conclusions: This unique microsphere-based system is a sensitive and efficient method to

simultaneously evaluate many **receptor**-coactivator interactions in a single assay volume. In addition, the system offers a powerful approach to study small molecule modulation of nuclear **receptor** binding. .COPYRGT. 2001 Wiley-Liss, Inc.

L9 ANSWER 4 OF 18 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2001673358 IN-PROCESS
DOCUMENT NUMBER: 21576087 PubMed ID: 11719067
TITLE: Juvenile hormone III-dependent conformational changes of the nuclear receptor ultraspiracle.
AUTHOR: Jones G; Wozniak M; Chu Y; Dhar S; Jones D
CORPORATE SOURCE: School of Biological Sciences, University of Kentucky, 40506, Lexington, KY, USA.
SOURCE: INSECT BIOCHEMISTRY AND MOLECULAR BIOLOGY, (2001 Dec) 32 (1) 33-49.
Journal code: BRE; 9207282. ISSN: 0965-1748.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20011126
Last Updated on STN: 20011126
AB The identification of potential endogenous or synthetic ligands for orphan **receptors** in the **steroid receptor** superfamily is important both for discerning endogenous regulatory pathways and for designing **receptor** inhibitors. The insect nuclear **receptor** Ultraspiracle (USP), an ortholog of vertebrate RXR, has long been treated as an orphan **receptor**. We have tested here the fit of terpenoid ligands to the JH III-binding site of monomeric and homo-oligomeric USP from. . . not control farnesol or JH III acid, and also specifically changed in conformation upon binding of JH III in a **fluorescence** binding assay. Juvenile hormone III binding caused intramolecular changes in **receptor** conformation, and stabilized the **receptor**'s dimeric/oligomeric quaternary structure. In both a radiometric competition assay and the **fluorescence** binding assay the synthetic JH III agonist methoprene specifically competed with JH III for binding to dUSP, the first demonstration of specific binding of a biologically active JH III analog to an insect nuclear **receptor**. The recombinant dUSP bound with specificity to a DR12 hormone response element in a gel shift assay. The same DR12. . . or T(3). The activity of JH III or JH III-like structures, but not structures without JH III biological activity, to **bind** specifically to dUSP and activate its conformational change, provide evidence of a terpenoid endogenous ligand for Ultraspiracle, and offer the. . .

L9 ANSWER 5 OF 18 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 2000270219 MEDLINE
DOCUMENT NUMBER: 20270219 PubMed ID: 10748001
TITLE: Orphan nuclear receptors constitutive androstane receptor and pregnane X receptor share xenobiotic and steroid ligands.
AUTHOR: Moore L B; Parks D J; Jones S A; Bledsoe R K; Consler T G; Stimmel J B; Goodwin B; Liddle C; Blanchard S G; Willson T M; Collins J L; Kliewer S A
CORPORATE SOURCE: Department of Molecular Endocrinology, Glaxo Wellcome Research and Development, Research Triangle Park, North Carolina 27709, USA.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 May 19) 275 (20) 15122-7.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200006
ENTRY DATE: Entered STN: 20000629
Last Updated on STN: 20000629
Entered Medline: 20000621

AB Xenobiotics induce the transcription of cytochromes P450 (CYPs) 2B and 3A through the constitutive androstane **receptor** (CAR; NR1I3) and pregnane X **receptor** (PXR; NR1I2), respectively. In this report, we have systematically compared a series of xenobiotics and natural **steroids** for their effects on mouse and human CAR and PXR. Our results demonstrate dual regulation of PXR and CAR by. . . both mouse and human PXR. Similarly, the PXR activator clotrimazole is a potent deactivator of hCAR. Using radioligand binding and **fluorescence** resonance energy transfer assays, we demonstrate that several of the compounds that regulate mouse and human CAR, including natural **steroids**, **bind** directly to the **receptors**. Our results suggest that CAR, like PXR, is a **steroid receptor** that is capable of recognizing structurally diverse compounds. Moreover, our findings underscore the complexity in the physiologic response to xenobiotics.

L9 ANSWER 6 OF 18 MEDLINE DUPLICATE 5
ACCESSION NUMBER: 2000240236 MEDLINE
DOCUMENT NUMBER: 20240236 PubMed ID: 10775644
TITLE: Cloning and characterization of bonnet monkey GnRH receptor.
AUTHOR: Santra S; Rao V S; Shanker Y G; Rao A J
CORPORATE SOURCE: Department of Biochemistry and Department of Molecular Reproduction, Development and Genetics, Indian Institute of Science, Bangalore 560 012, India.
SOURCE: MOLECULAR HUMAN REPRODUCTION, (2000 May) 6 (5) 415-21. Journal code: CWO; 9513710. ISSN: 1360-9947.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000810
Last Updated on STN: 20000810
Entered Medline: 20000725

AB . . . plays an important role in the reproductive processes of both males and females. It is synthesized by the hypothalamus and **binds** to a specific **receptor** on the pituitary to bring about the release of the gonadotrophins, lutineizing hormone and follicle stimulating hormone, which in turn bring about the release of the gonadal **steroids**. Although the structure of the GnRH **receptor** (GnRHR) has been elucidated from a number of sources, no information is available about the **receptor** from the non-human primate species. Here we report the cloning and characterization of the **receptor** from the pituitary of the bonnet monkey. Antiserum to a bacterially expressed recombinant fragment was used in Western blot analysis and **fluorescence** microscopy to demonstrate the presence of GnRHR in both human and monkey placentae and pituitary.

L9 ANSWER 7 OF 18 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:112498 CAPLUS
DOCUMENT NUMBER: 128:176476
TITLE: A method for quantitating competitive binding of molecules to steroid hormone receptors utilizing fluorescence polarization
INVENTOR(S): Bolger, Randall E.; Ervin, Kerry M.; Lowery, Robert G.; Checovich, William J.
PATENT ASSIGNEE(S): Panvera Corp., USA; Burke, Thomas, J.
SOURCE: PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9805962	A1	19980212	WO 1997-US13538	19970801

RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1996-23034 19960802
AB The system comprises mixing a **fluorescence**-emitting compd. that **binds** to the **steroid** hormone **receptors**, particularly the estrogen **receptor**, in a soln. contg. the **steroid** hormone **receptors**. Then, measuring the **fluorescence** polarization of the soln. Subsequently, incubating the soln. with at least one mol. that may compete with the compd. for interaction with the **steroid** hormone **receptors**. Measuring the **fluorescence** polarization of the soln. again. Finally, comparing the **fluorescence** polarization measurements to quantify any competitive interaction. A **fluorescence**-emitting compd. such as a **fluorescence**-emitting hormone can be used in combination with a fluorophore covalently coupled to an oligonucleotide to study how hormone and oligonucleotide binding to the hormone **receptor** are affected by each other.

L9 ANSWER 8 OF 18 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 1998343574 MEDLINE
DOCUMENT NUMBER: 98343574 PubMed ID: 9679980
TITLE: Expression and location of Hsp70/Hsc-binding anti-apoptotic protein BAG-1 and its variants in normal tissues and tumor cell lines.
AUTHOR: Takayama S; Krajewski S; Krajewska M; Kitada S; Zapata J M; Kochel K; Knee D; Scudiero D; Tudor G; Miller G J; Miyashita T; Yamada M; Reed J C
CORPORATE SOURCE: The Burnham Institute, La Jolla, California 92037, USA.
CONTRACT NUMBER: CA67329 (NCI)
SOURCE: CANCER RESEARCH, (1998 Jul 15) 58 (14) 3116-31.
Journal code: CNF; 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980820
Last Updated on STN: 19980820
Entered Medline: 19980807
AB . . . apoptosis and interacts with several types of proteins, including Bcl-2 family proteins, the kinase Raf-1, certain tyrosine kinase growth factor **receptors**, and **steroid** hormone **receptors**, possibly by virtue of its ability to regulate the Hsp70/Hsc70 family of molecular chaperones. Two major forms of the human. . . another site involving an ATG codon. All three isoforms of human BAG-1 (BAG-1, BAG-1M, and BAG-1L) retained the ability to **bind** Hsc70. Subcellular fractionation and immunofluorescence confocal microscopy studies indicated that BAG-1L often resides in the nucleus, consistent with the presence. . . organelles resembling mitochondria, consistent with the reported interaction of BAG-1 with Bcl-2 and related proteins. Furthermore, experiments using a green **fluorescence** protein (GFP)-BAG-1 fusion protein demonstrated that overexpression of Bcl-2 in cultured cells can cause intracellular redistribution of GFP-BAG-1, producing a. . .

L9 ANSWER 9 OF 18 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 1998400437 MEDLINE
DOCUMENT NUMBER: 98400437 PubMed ID: 9731711
TITLE: Functional antagonism of gonadal steroids at the 5-hydroxytryptamine type 3 receptor.
AUTHOR: Wetzel C H; Hermann B; Behl C; Pestel E; Rammes G; Zieglgansberger W; Holsboer F; Rupprecht R
CORPORATE SOURCE: Max Planck Institute of Psychiatry, Munich, Germany.
SOURCE: MOLECULAR ENDOCRINOLOGY, (1998 Sep) 12 (9) 1441-51.
Journal code: NGZ; 8801431. ISSN: 0888-8809.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981125

AB **Steroid** hormone action involves binding to cognate intracellular **receptors** that, in turn, **bind** to respective response elements and thus modulate gene expression. The present study shows that the gonadal **steroids**, 17beta-estradiol and progesterone, may also act as functional antagonists at the 5-hydroxytryptamine type 3 (5-HT3) **receptor** in whole-cell voltage-clamp recordings of HEK 293 cells stably expressing the 5-HT3 **receptor**. Functional antagonistic properties at this ligand-gated ion channel could also be shown for 17alpha-estradiol, 17alpha-ethinyl-17beta-estradiol, mestranol, R 5020, testosterone, and allopregnanolone but not for pregnenolone sulfate and cholesterol. An antagonism at the 5-HT3 **receptor** could further be observed with the aromatic alcohol 4-dodecylphenol but not with phenol or ethanol. Thus, the modulation of 5-HT3 **receptor** function by **steroids** or alcohols is dependent on their respective molecule structure. The antagonistic action of **steroids** at the 5-HT3 **receptor** is not mediated via the serotonin binding site because the **steroids** did not alter the binding affinity of [3H]GR65630 to the 5-HT3 **receptor**, and kinetic experiments revealed a quite different response pattern to 17beta-estradiol when compared with the competitive antagonist metoclopramide. BSA-conjugated gonadal **steroids** labeled with fluorescein isothiocyanate bound to membranes of HEK 293 cells expressing the 5-HT3 **receptor** in contrast to native HEK 293 cells. However, there was no dose-dependent displacement of the binding of gonadal **steroids** to membranes of cells expressing the 5-HT3 **receptor** in binding experiments or **fluorescence** studies. Thus, gonadal **steroids** probably interact allosterically with the 5-HT3 **receptor** at the **receptor**-membrane interface. The functional antagonism of gonadal **steroids** at the 5-HT3 **receptor** may play a role for the development and course of nausea during pregnancy and of psychiatric disorders.

L9 ANSWER 10 OF 18 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:476304 CAPLUS
DOCUMENT NUMBER: 127:105220
TITLE: Monitoring DNA binding molecules in living cells containing a steroid receptor response element array using a fluorescent chimeric protein of the steroid receptor
INVENTOR(S): Htun, Han; Hager, Gordon L.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA;
Htun, Han; Hager, Gordon L.
SOURCE: PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9720931	A1	19970612	WO 1996-US19516	19961206
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2239951	AA	19970612	CA 1996-2239951	19961206
AU 9712834	A1	19970627	AU 1997-12834	19961206

PRIORITY APPLN. INFO.: US 1995-8373 P 19951208
WO 1996-US19516 W 19961206

AB A method of screening for a compd. that **binds** to a selected nucleic acid is provided that comprises contacting a compd. fluorescently labeled by a fluorescent protein with a cell having a plurality of copies of the nucleic acid in an array such that the nucleic acid can be directly detected when bound by fluorescently labeled compd. The location of **fluorescence** within the cell is detected such that **fluorescence** aggregated at the site of the nucleic acid array indicates a compd. that **binds** to the selected nucleic acid. In particular compds. such a transcription factor can be screened. Reagents for such method are provided including a mammalian cell having a plurality of **steroid receptor** response elements in an array such that the response element can be directly detected when bound by fluorescently labeled **steroid receptor** and a chimeric protein comprising a fluorescent protein fused to a **steroid receptor**. Thus, a chimeric protein is constructed comprising a 27-kDa green fluorescent protein (GFP, from Aequorea victoria) and fused by a (Gly-Ala)5 peptide linker to the N-terminal second residue of rat glucocorticoid **receptor** (GR). Improved **fluorescence** is achieved by using a GFP variant contg. a serine-65 to threonine substitution, which increases the efficiency of formation of the GFP chromophore, and a GR variant contg. a cysteine-656 to glycine mutation has higher affinity for its ligand than endogenous **receptor**. A mammalian cell line named 3134 was derived by transfection of murine mammary carcinoma line C127 with a plasmid contg. 3 functional segments: (a) the bovine papilloma virus 69% transforming fragment serving as a replicon in mammalian cells; (b) mouse mammary tumor virus (MMTV) LTR is a **steroid** responsive promoter and contains the GR binding sites; and (c) the Ha-v-ras gene is a transforming oncogene and serves as a reporter for the MMTV promoter. The MMTV LTRs are organized in a head-to-tail tandem array of .apprx.200 copies, and since each promoter sequence contains 4 GR binding sites, the complete array contains 100 GR binding sites. This cell is used to visualize directly the interaction between the fluorescent, chimeric GR and its binding site in chromatin in living cells. The system allows screening for DNA-binding ligands that (1) activate gene targeting by **steroid receptor**, (2) activate translocation of the **steroid receptor** to the nucleus, (3) are antagonists or agonists of the **steroid receptor**.

L9 ANSWER 11 OF 18 MEDLINE DUPLICATE 8
ACCESSION NUMBER: 1998008920 MEDLINE
DOCUMENT NUMBER: 98008920 PubMed ID: 9344655
TITLE: Chromosomal mapping of the human and murine orphan receptors ERRalpha (ESRRA) and ERRbeta (ESRRB) and identification of a novel human ERRalpha-related pseudogene.

AUTHOR: Sladek R; Beatty B; Squire J; Copeland N G; Gilbert D J; Jenkins N A; Giguere V

CORPORATE SOURCE: Royal Victoria Hospital, Department of Biochemistry, McGill University, 687 Pine Avenue West, Montr-eal, Quebec, H3A 1A1, Canada.

SOURCE: GENOMICS, (1997 Oct 15) 45 (2) 320-6.
Journal code: GEN; 8800135. ISSN: 0888-7543.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-AF022222; GENBANK-U85258; GENBANK-X51416

ENTRY MONTH: 199802

ENTRY DATE: Entered STN: 19980224
Last Updated on STN: 19980224
Entered Medline: 19980212

AB The estrogen-related **receptors** ERRalpha and ERRbeta (formerly ERR1 and ERR2) form a subgroup of the **steroid**/thyroid/retinoid **receptor** family. ERRalpha and ERRbeta are homologous to the estrogen **receptor** and **bind** similar DNA targets; however, they are unable to activate gene transcription in response to estrogens. We have used interspecific backcross analysis to map the murine Estrra locus to chromosome 19 and Estrrb to mouse chromosome 12. Using **fluorescence** in situ hybridization, we have mapped the human ESRRA gene to chromosome 11q12-q13 and the human ESRRB gene to chromosome. . chromosome 13q12.1. To our knowledge, this represents the first report of a pseudogene associated with a member of the nuclear **receptor** superfamily.

L9 ANSWER 12 OF 18 MEDLINE DUPLICATE 9

ACCESSION NUMBER: 86221226 MEDLINE

DOCUMENT NUMBER: 86221226 PubMed ID: 3011559

TITLE: Sex steroid and prostaglandin interactions upon the purified rat myometrial plasma membranes.

AUTHOR: Deliconstantinos G; Fotiou S

SOURCE: MOLECULAR AND CELLULAR ENDOCRINOLOGY, (1986 May) 45 (2-3) 149-56.
Journal code: E69; 7500844. ISSN: 0303-7207.

PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198607

ENTRY DATE: Entered STN: 19900321
Last Updated on STN: 19900321
Entered Medline: 19860710

AB . . . with myometrial plasma membranes (MPM) at a concentration of 1 X 10(-6) M for 1 h at 37 degrees C, **bind** into MPM at pmolar concentrations. Unlabeled **steroids** inhibited [3H]PGE2 and [3H]PGF2 alpha binding to MPM in a dose-dependent manner. Membrane-bound and free **steroids** or PGs were found to be essentially unchanged under the present incubation conditions. Ca2+ ions up to 10 mM increased **steroid** binding into MPM. Molecular interactions between **steroids** and MPM were assessed by measuring the steady-state **fluorescence** polarization of 1,6-diphenyl-1,3,5-hexatriene (DPH), and by estimating the changes in the allosteric properties of MPM-bound (Na+ + K+)ATPase by fluoride (F-). **Steroids** appear to increase the MPM fluidity, evaluated through changes in the Hill coefficient for MPM-bound (Na+ + K+)ATPase by F- and by the **fluorescence** polarization method. Binding of sex **steroids** to MPM increased the membrane fluidity and decreased the binding of the uterus stimulatory PGs by membrane **receptors**. These studies provide a basis for

postulating that a 'non-genomic' mechanism of sex **steroids** induces reduction of uterine contractions.

L9 ANSWER 13 OF 18 MEDLINE DUPLICATE 10
ACCESSION NUMBER: 85201339 MEDLINE
DOCUMENT NUMBER: 85201339 PubMed ID: 2986819
TITLE: Biochemical and histochemical analysis of steroid hormone binding sites in human primary breast cancer.
AUTHOR: Janssens J P; Pylyser K; Bekaert J; Roelens J; Stuyck J; Dekeyser L J; Lauweryns J M; De Loecker W
SOURCE: CANCER, (1985 Jun 1) 55 (11) 2600-11.
Journal code: CLZ; 0374236. ISSN: 0008-543X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198506
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850626

AB Mammary carcinoma tissue from 514 primary breast cancer patients were all biochemically and histochemically analyzed for both estrogen **receptors** and progesterone **receptors**. The dextran-coated charcoal (DCC) method measured the ER and PR as defined by Scatchard analysis, ligand competition experiments and target organ specificity. The ligands, estradiol-6-carboxymethyloxime-BSA-fluoresceine isothiocyanate and hydroxyprogesteronehemisuccinate-BSA-tetramethylrhodamine isothiocyanate, used for histochemistry, did not **bind** to either ER or PR and were mainly bound to the membrane fraction of isolated breast cancer cells. **Fluorescence** was not specifically inhibited by estrogens or progestogens. In addition, "estrogenic" always coincided with "progestogenic" **fluorescence**. The binding of the fluoresceine compounds to tissue slides depended on the large **steroid** hormone substitution on the bovine serum albumin molecule. Clinical parameters, known to be related to ER and PR did not correlate with the histochemical results. The observations indicated the impossibility of specific **steroid receptor** detection by the histochemical method. Therefore, up to the present, evaluation of hormone dependency and prognosis in human breast cancer. . .

L9 ANSWER 14 OF 18 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 85142145 EMBASE
DOCUMENT NUMBER: 1985142145
TITLE: Biochemical and histochemical analysis of steroid hormone binding sites in human primary breast cancer.
AUTHOR: Janssens Ph. J.; Pylyser K.; Bekaert J.; et al.
CORPORATE SOURCE: Afdeling Biochemie, Katholieke Universiteit te Leuven, B-3000 Leuven, Belgium
SOURCE: Cancer, (1985) 55/11 (2600-2611).
CODEN: CANCAR
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
016 Cancer
009 Surgery
005 General Pathology and Pathological Anatomy
003 Endocrinology
006 Internal Medicine
029 Clinical Biochemistry
LANGUAGE: English

AB Mammary carcinoma tissue from 514 primary breast cancer patients were all biochemically and histochemically analyzed for both estrogen **receptors** and progesterone **receptors**. The dextran-coated

charcoal (DCC) method measured the ER and PR as defined by Scatchard analysis, li[redacted]d competition experiments and [redacted]rget organ specificity. The ligands, estradiol-6-carboxymethyloxime-BSA-fluoresceine isothiocyanate and hydroxyprogesteronehemisuccinate-BSA-tetramethylrhodamine isothiocyanate, used for histochemistry, did not **bind** to either ER or PR and were mainly bound to the membrane fraction of isolated breast cancer cells. **Fluorescence** was not specifically inhibited by estrogens or progestogens. In addition, 'estrogenic' always coincided with 'progestogenic' **fluorescence**. The binding of the fluoresceine compounds to tissue slides depended on the large **steroid** hormone substitution on the bovine serum albumin molecule. Clinical parameters, known to be related to ER and PR did not correlate with the histochemical results. The observations indicated the impossibility of specific **steroid receptor** detection by the histochemical method. Therefore, up to the present, evaluation of hormone dependency and prognosis in human breast cancer. . .

L9 ANSWER 15 OF 18 MEDLINE DUPLICATE 11
ACCESSION NUMBER: 84137204 MEDLINE
DOCUMENT NUMBER: 84137204 PubMed ID: 6366089
TITLE: Implications of subcellular steroid binding sites in endometrial cancer, determined by an immunofluorescent steroid-antibody technique and biochemical assay.
AUTHOR: Tamaya T; Kimura J; Tsurusaki T; Kato Y; Fujimoto J; Okada H
SOURCE: NIPPON SANKA FUJINKA GAKKAI ZASSHI. ACTA OBSTETRICA ET GYNAECOLOGICA JAPONICA, (1984 Jan) 36 (1) 113-8. Journal code: INR; 7505749. ISSN: 0300-9165.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198403
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19840326

AB A discrepancy has been found between the progestogen level necessary for treatment of endometrial cancer and the **steroid receptor** level detected for the response indicator. Therefore the relationships between the **steroid** binding quantity detected biochemically and the **steroid** reactivity determined immunofluorescently was evaluated subcellularly in the endometrial cancers. Estradiol-17 beta and progesterone **fluorescences** were not always related to the classical **steroid receptor** binding quantities. These two **steroids** bound to the nuclear components directly, but heterogeneously. In the biochemical method using fractionated dispersed cancer cells, cellular heterogeneity of the **steroid receptor** mechanism in a given endometrial cancer tissue was proved. **Steroid fluorescence** was not related to the **steroid-receptor** complex quantity in the normal endometrial nucleus. This suggests that the binding of **steroid** antibody to the **steroid-receptor** bound already to the nucleus seems to be inhibited due to steric hindrance. Therefore the nuclear **steroid fluorescence** did not always give the nuclear **steroid-receptor** complex quantity. These results indicate heterogeneity in the estrogen and progestogen **receptor** mechanism in endometrial cancer, when studied by the biochemical and immunofluorescent techniques, and that these **steroids bind** to the nucleus directly and may influence the nuclear mechanism. Therefore, in endometrial cancer progestogen does not always have a therapeutic effect through the progestogen **receptor** and does not affect the therapeutic effect on any of the cells.

L9 ANSWER 16 OF CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1984:448844 CAPLUS
DOCUMENT NUMBER: 101:48844
TITLE: On the use of poly- and monoclonal antibodies in studies on the structure and function of the glucocorticoid receptor
AUTHOR(S): Gustafsson, Jan Aake; Okret, Sam; Wikstroem, Ann Charlotte; Andersson, Birger; Radojcic, Maja; Wrange, Oerjan; Sachs, Wendy; Doupe, Allison J.; Patterson, Paul H.; et al.
CORPORATE SOURCE: Dep. Med. Nutr., Karolinska Inst., Huddinge, 141 86, Swed.
SOURCE: Nobel Symp. (1983), 57(Steroid Horm. Recept.: Struct. Funct.), 355-88
CODEN: NOSYBW; ISSN: 0346-8313
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Polyclonal antibodies against the glucocorticoid **receptor** (GR) were used to describe the existence of a nonliganded form of GR which occurs as a monomer even in the absence of molybdate. A model was presented for the ligand binding and activation of this GR in the presence and absence of molybdate. In the absence of molybdate the nonliganded oligomeric GR 1st dissocs. and then **binds** the **steroid**; whereas, in presence of molybdate the **steroid** is bound by the GR and the oligomeric liganded GR then dissocs. The polyclonal antibodies and **fluorescence** were also used to detect GR in adrenergic neurons and in the rat superior cervical ganglia nuclei. GR were also detected in fetal rat adrenal medullary cells in culture. The prepn. of 10 monoclonal antibodies against the rat GR was described. These were used for the immunohistochem. localization of GR in the central nervous system. GR immunoreactive neurons were detected in the diencephalon and some of these resemble corticosterone-concg. neurons previously obsd. However, the no. of GR immunoreactive neurons far exceeds the no. of corticosterone-concg. neurons. Highly pos. GR nerve cell nuclei were demonstrated in the paraventricular, periventricular, and mediobasal hypothalamic neuron system for the 1st time. Apparently, the highest GR immunoreactivity is found in areas involved in the regulation of secretion of pituitary hormones, esp. CRF [9015-71-8], suggesting the involvement of glucocorticoids in the control regulation of cRF secretion.

L9 ANSWER 17 OF 18 MEDLINE DUPLICATE 12

ACCESSION NUMBER: 81211938 MEDLINE
DOCUMENT NUMBER: 81211938 PubMed ID: 7238414
TITLE: Heterogeneity of nuclear estrogen-binding sites in the rat uterus: a simple method for the quantitation of type I and type II sites by [3H]estradiol exchange.
AUTHOR: Markaverich B M; Williams M; Upchurch S; Clark J H
CONTRACT NUMBER: CA-20605 (NCI)
CA-26112 (NCI)
HO-08436 (NHLBI)
SOURCE: ENDOCRINOLOGY, (1981 Jul) 109 (1) 62-9.
Journal code: EGZ; 0375040. ISSN: 0013-7227.
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AB . . . causes the activation or stimulation of secondary nuclear estrogen-binding sites (type II) in the uterus which can interfere with

estrogen **receptor** (type I) measurement. Earlier reports from our laboratory h[redacted] shown that quantitation of ty[redacted] I sites in the presence of. . . separately quantitate both nuclear estrogen-binding sites using a single concentration of [3H]estradiol. Since the nuclear type II site does not **bind** [3H]estradiol in the presence of reducing agent, type I sites can be easily quantitated by incubating nuclei (37 C for. . . by incubating nuclei in Tris-EDTA buffer under conditions (4 C for 60 min) which do not measure occupied nuclear estrogen **receptor**. Therefore, by using the appropriate buffer system, type I and type II sites can be easily separated in mixed binding systems. In addition, we also demonstrate that Nafoxidine does not **bind** to the nuclear type II site. Therefore, it can be used as a competitive inhibitor of [3H]estradiol binding to type. . . the measurement of type II sites without interference from type I sites. These techniques should be applicable to autoradiographic or **fluorescence** studies which cannot discriminate between **steroid** binding to these two classes of nuclear estrogen-binding sites.

L9 ANSWER 18 OF 18 CAPLUS COPYRIGHT 2002 ACS
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ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB **Fluorescence** spectrometry data by Tyulmenkov and Klinge (Arch. Biochem. Biophys. 2000, 381, 135-142) suggest the presence of a second binding site in both subtypes ER.alpha. and ER.beta. of the estrogen **receptor** (ER). A cavity previously described as a solvent channel was located in close proximity to the **steroid** binding site of both ER subtypes. Derivs. of a tetrahydrochrysene (THC) compd., speculated in the literature to **bind** to a second binding site, were docked successfully in the second sites identified. However, computation of accurate interaction scores indicates preferred binding to the **steroid** binding site over the second binding site of both ER.alpha. and ER.beta. for all THC derivs. Therefore, binding to this second site is probably not the reason the THC derivs. are agonists on ER.alpha. and antagonists on ER.beta.. Most likely, the smaller **steroid** binding site of ER.beta. compared to ER.alpha. and/or the apparent larger flexibility of helix 12 of ER.beta. make ER.beta. more readily adopt an antagonist conformation.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	72.90	73.11

DISCOUNT AMOUNTS [redacted]R QUALIFYING ACCOUNTS)	S[redacted]E FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-4.34	-4.34

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